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Dr. J. Hamilton Crawford, Brooklyn, *Editor*

Dr. Charles A. Poindexter, New York, *Associate Editor*

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THE VENTRICULAR COMPLEX IN MULTIPLE PRECORDIAL LEADS

PART I

The practice of taking a single precordial lead and calling it Lead IV has fostered the idea that there are no fundamental differences between leads from the precordium and leads from the extremities. This notion has led to the conclusion that the standardization of the former must be founded solely upon an extensive experience with each and every method of taking such leads that has been devised. It has, however, been clearly demonstrated that precordial leads of the kind now popular are really semidirect leads from the anterior surface of the heart and faithfully record the potential variations of the part of the ventricular surface nearest to the exploring electrode. They may be regarded as clinical substitutes for and yield information similar to unipolar direct leads of the kind introduced by Lewis and Rothschild[1] in their study of the spread of the excitatory process over the dog's heart. This makes them capable of detecting local lesions of the anterior wall of the heart which produce no significant alterations in the ventricular deflections of limb leads.

It would seem, then, that the best possible precordial leads are those which most faithfully reproduce the potential variations of the part of the epicardial surface nearest the precordial electrode. It is also clear that the number of such leads taken must be great enough to give adequate information regarding the potential variations at the surfaces of both ventricles. In our laboratory the six leads recognized by the Committee of the American Heart Association on the Standardization of Precordial Leads[2] and designated Leads V_1, V_2, V_3, V_4, V_5 and V_6 are always taken and, in addition, leads from the tip of the ensiform cartilage (V_E), from the left posterior axillary line (V_7), from the left scapular line (V_B), and from the ventricular levels of the esophagus are often employed. The last are particularly useful in the diagnosis of infarction of the posterior wall of the left ventricle.

In experiments on dogs we have compared the ventricular complexes obtained by precordial leads of this sort with those of unipolar direct leads from different parts of the anterior surfaces of the right and left ventricles. In this way we have demonstrated in right and left bundle branch block, in infarction of the right and in infarction of the left ventricle, in bundle branch block complicated by infarction of the left ventricle, and when the heart is normal that there is a close resemblance between the potential variations of the different parts of the precordium and the potential variations of the subjacent ventricular surface.

In CR, CL, and CF leads, in which the exploring electrode is paired with an electrode on one of the extremities, the potential variations of the precordium are not recorded in pure form, but are distorted by the potential variations of the limb electrode. The magnitude of this distortion is a measure of the extent to which the purpose of placing one electrode on the precordium has been defeated by pairing this electrode with another that is not strictly indifferent. It was to avoid such distortion that a central terminal connected through resistances of 5000 ohms to electrodes on the right arm, left arm, and left leg was introduced as the reference point[3]. The potential of such a terminal is at every instant the mean potential of the three extremity electrodes, which is nearly constant. This is the method which we use and it has been amply demonstrated that it is the best simple method of avoiding the distortion in question. Studies by Goldberger[4] and observations made in this laboratory indicate that conductors of negligible resistance can be substituted for the resistances of 5000 ohms used to connect the central terminal to the electrodes on the extremities without materially altering the form of the curve obtained.

A single precordial lead is unsatisfactory because it can give information concerning the potential variations of only a small part of the anterior ventricular surface, and also because there is often a good deal of uncertainty as to what particular fraction of this surface is mainly responsible for the potential variations recorded. When multiple precordial leads are taken these difficulties are avoided and the changes in the character of the ventricular deflections as the exploring electrode is moved across the precordium from right to left give clues to the analysis of the ventricular complex that are indispensable.

It is evident that the interpretation of the different QRS components of unipolar precordial leads must be based upon study of the analagous deflections of unipolar direct leads taken in animal experiments. In such leads the peak of the principal upward deflection (R or R′) marks the onset of the intrinsic deflection which signals the activation of the subepicardial muscle beneath the exploring electrode. When this peak occurs early in the QRS interval the R wave is usually small and is followed by a deep S wave; when it occurs late in the QRS interval the R wave is large and S is small or absent. The R wave is often preceded by a Q deflection when it is late, seldom when it is early. In other words the form of the QRS complex is largely determined by the time of activation of the muscle beneath the exploring electrode in relation to the spread of the excitatory process over the ventricular muscle as a whole.

In normal subjects leads from the right side of the precordium yield a small initial R wave which reaches its peak early in the QRS interval and a deep S component. The QRS complex is of this form because the cardiac impulse spreading from within outward reaches the epicardial surface of the thin free wall of the right ventricle much earlier than the

outer surface of the thick-walled left ventricle. In leads from the left side of the precordium the R deflection is much larger and reaches its apex late in the QRS interval and S is small or absent. In these leads there is often a small Q deflection. In the corresponding direct leads this deflection is due to the transmission of the potential of the ventricular cavity, which is negative throughout the QRS interval, to the exploring electrode during the time when no part of the wall between this electrode and the cavity has yet passed into the active state. In other words, a Q deflection occurs when the inner layers of the ventricular wall adjacent to the exploring electrode become active late in comparison with the other regions of subendocardial muscle. The ventricular cavity becomes negative as soon as any substantial part of the muscle bounding it is activated. In the case of the normal heart in which both ventricular cavities are negative throughout the QRS interval, it is clear that a positive deflection (R or R') in a unipolar epicardial or precordial lead must be due to forces generated in that part of the ventricular wall subjacent to the exploring electrode while negative deflections (Q and S) must be attributed to the electrical forces arising in other parts of the ventricular muscle.

The ventricular complexes obtained by leading from the central part of the precordium are intermediate in form between those obtained from points farther to the right and those obtained from points farther to the left. The location and width of this transitional zone are quite variable. In most normal subjects its center is close to the point from which the third precordial lead is taken, but it may be either nearer or farther from the sternum. The factors which determine its position and extent are still obscure.

In the precordial electrocardiograms of normal adult subjects the T wave is occasionally inverted in lead V_1 but not in the other leads. On the average the peak of R is about 0.02 sec. earlier in the leads from the right side of the precordium than in the leads from the left.

Ventricular hypertrophy. In patients with left ventricular enlargement due to arterial hypertension or lesions of the aortic valve precordial leads yield ventricular complexes differing from those obtained in normal subjects in the following respects: (a) The voltage of the largest QRS deflection is on the average much greater. (b) The QRS interval is often increased to 0.10 or 0.11 second. (c) In the leads from the right side of the precordium the initial R deflection is abnormally small and may be absent. (d) The transitional zone is shifted to the left. (e) In the leads from the left side of the precordium R, and often Q as well, are larger; the peak of R occurs later in the QRS interval, and the T wave is usually inverted. It should be noted that inversion of *T* waves in these leads may be due to digitalis if this drug has been given.

In cases of left ventricular enlargement with these characteristic changes in the precordial electrocardiogram the standard leads may exhibit ventricular deflections of several types. Left axis deviation with inversion of T_1 is the common pattern observed. It occurs when the long axis of the heart is relatively horizontal so that the potential variations of the left ventricular surface are transmitted to the left arm. In occasional electrocardiograms there are abnormally large R waves and inverted T waves in all leads and in rare instances right axis deviation. Curves of these types are found when the long axis of the heart is more vertical and the potential variations of the left ventricular surface are transmitted to the left leg.

In cases of great right ventricular hypertrophy the precordial curves are opposite in form to those obtained in left ventricular hypertrophy, possibly because the right ventricular wall is thicker than the left. In the leads from the right side of the precordium the R waves are very large, a Q deflection is often present, S is small or absent, and T is frequently inverted. The ventricular complexes of the leads from the left side of the precordium are like those normally obtained from the right side; R is very small, S very large, Q absent, and T upright. These precordial curves are somewhat like those seen in right bundle branch block but the latter show a longer QRS interval, bifid or notched R waves in the leads from the right side of the precordium and usually narrow R waves of nearly normal voltage followed by broad slurred S waves in leads from the left side of the precordium. When the right ventricular hypertrophy is less pronounced the precordial curves are less characteristic but are transitional in form between those described and those obtained in normal subjects.

In cases in which the precordial curves are characteristic of pronounced right ventricular hypertrophy the limb leads display right axis deviation if the heart is horizontally placed and large S waves in all leads if its long axis is relatively vertical. It is possible that in extremely rare cases left axis deviation occurs but we have not seen an unquestionable case of this sort.

F. D. Johnston, M.D.
F. F. Rosenbaum, M.D.
F. N. Wilson, M.D.
Ann Arbor, Michigan

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